FEB 1 1 1944

Volume V FEBRUARY 1944 Number 2

Medical Library

DISEASES OF THE NERVOUS SYSTEM

A PRACTICAL JOURNAL ON PSYCHIATRY AND NEUROLOGY

PUBLISHED MONTHLY for the PHYSICIAN in the GENERAL PRACTICE OF MEDICINE

CONTENTS:

THE ORGANIC AND PHYSIOLOGIC FACTORS IN MENTAL DEFECT (Part II) ..... Page 37
Harold D. Palmer, M.D., Philadelphia, Pa.

THE HISTORY OF PSYCHIATRY ..... Page 52
Roy W. Menninger, Topeka, Kansas

A FURTHER REPORT ON THE USE OF SHOCK THERAPY (Results in 1302 Cases) ..... Page 56
John H. Taylor, M.D., Trenton, New Jersey

NEUROLOGICAL CLINICAL PATHOLOGICAL CONFERENCE ..... Page 57

BOOK REVIEW. SANITARIA

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800 AVENUE B., GALVESTON, TEXAS

BUSINESS OFFICES
PHYSICIANS POSTGRADUATE PRESS
500 NORTH DEARBORN ST., CHICAGO 10, ILLINOIS

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PHYSICIANS POSTGRADUATE PRESS

DISEASES of the NERVOUS SYSTEM

(PUBLISHED MONTHLY)

A PRACTICAL JOURNAL ON PSYCHIATRY AND NEUROLOGY

The Organic and Physiologic Factors in Mental Defect*

HAROLD D. PALMER, M.D.
Philadelphia, Pennsylvania

PART II

IV. *Mental Defect Due to Developmental Structural Pathology in the Central Nervous System*

A certain percentage of mental defects result from absence or incomplete formation of portions of the nervous system. In some instances there is evidence indicating that development has occurred up to a certain point and then destruction has taken place. Ford points out that it is impossible to discern whether the etiology is early intrauterine disease or originally defective germ plasm since defective nervous systems may react excessively to maternal intoxications, infections, and mild trauma, thus producing a picture of complicated interactions. Certain developmental defects are, however, clearly hereditary in nature and quite properly belong under the heading "Mental defect due to hereditary or as yet unknown influences." Others must be placed in another group, with the full knowledge that their classification is based on direct and obvious rather than fundamental causation and with the expectation that they will not remain permanently under the above heading.

A. *Porencephaly*—True porencephaly is a localized developmental defect usually in the form of a funnel shaped cyst or opening in the brain surface. The cyst is covered with pia-arachnoid and opens into the ventricular system. Ford differentiates a true and a false porencephaly, the latter being the result of injury or intrauterine disease. The cysts may vary in size from small symptomless outpouchings of the ventricle to cysts which replace nearly all of the brain tissue of an entire hemisphere. With the larger cysts the symptoms of hemiplegia, aphasia, Jacksonian convulsions and mental deficiency may be apparent in early infancy while with the smaller cysts the symptoms may appear as late as the sixth or eighth year, or in rare instances not at all. The chief symptoms, of course, depend upon the area involved. Usually the cysts occupy the frontal portion of the brain involving the motor area and giving rise to motor symptoms such as hemiplegia, convulsions, etc. The air encephalogram will show the exact localization, and surgical removal of the roof of the cyst is the only method of treatment. This can be expected to relieve the convulsive seizures but the hemiplegia remains unaffected. Occasionally a cyst of considerable size can exist without serious impairment of intellectual development.

Case example of porencephaly with mental deficiency—A boy, aged 17 years, six months, with a history of normal birth after a normal pregnancy and normal appearance after birth, had convulsions when aged 18 months. He was treated by x-ray for thymus gland enlargement without benefit and by a number

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of Christian Science healers, also without benefit. He was destructive and constantly noisy, cutting up sheets, groaning, screeching, etc. At 12 years of age he had extremely severe and frequent convulsions. He was examined by Dr. Dandy in Baltimore, who found a large scotoma. X-rays showed a large porencephalic cyst in the left occipital lobe. He was operated on by Dr. Dandy in 1938 and the entire left occipital lobe was excised. A cyst 2x5 cm. was found, fibrous tissue was encountered and cysts of the glomus of the choroid plexus were visible and coagulated. No convulsions have occurred since the operation. The patient is difficult, restless and noisy but is capable of performing a few useful tasks. Psychometric findings at age 17 were as follows:

Stanford-Binet

Mental Age	10 years, 4 months
I. Q.	65

Shipley-Hartford Scale

Mental Age	9 years, 8 months
I. Q.	60

Thorndike-McCall Reading Scale

Reading Age	10 years, 1 month
R. Q.	63

Carl Hollow Square

Mental Age	15 years, 4 months
I. Q.	96

B. *Anencephaly and other forms of lack of cerebral structure*—Anencephaly is a grave developmental defect which is fortunately incompatible with more than a few weeks of life. According to Ford, the forebrain and midbrain are absent and are represented by a spongy mass of incompletely differentiated tissue known as the area cerebro-vasculosa. The hindbrain is more or less complete. Various other extensive developmental defects, such as hemicephaly, exencephaly, meningoencephalocele and complete rachischisis, are the result of defective closure of midline structures in embryonic life. They, like anencephaly, present no problem of management of mental defect, since life is only a matter of days or at most a few weeks.

C. *Microcephaly*, or failure of the cranium to attain normal size, with consequent faulty development of cerebral tissue is caused by primordial pathogenic factors, such as toxins, alcoholism, syphilis and nutritional toxemia, acting on the embryo itself or on the already altered germinal cells before conception. It may also be caused by x-ray treatment of the mother during pregnancy, according to a report published by Murphy in 1928. Organic symptoms of this disease are various, including usually some evidence of neurological disorder and in all cases a severe degree of mental deficiency. The behavior of microcephalics is characterized by lack of inhibitory control and by extreme restlessness similar to that observed in severe cases of cerebral damage. In an exhaustive study of two cases of low grade idiocy associated with microcephaly, Benda discusses the anatomy and histopathology of the condition. In one case there was developmental arrest with microgyria interna and macrogyria. In its degree of development the brain of this case, aged 10 years, 1 month, corresponded to that of a six months' fetus. The second case showed a cystic degeneration of white matter, indicated by the general history to have been caused by prolonged asphyxia at birth. Both cases showed microhydrocephaly, or the presence of excessive fluid in a microcephalic cranium. This can be determined by the air encephalogram, and presents a syndrome of small head, early closure of the fontanelles, defective vision, spasticity and enlarged ventricles, as well as mental defect. In a study of 13 cases of this syndrome, McClelland concluded that the condition was prenatal in origin, and was unable to find any causative agency. This form of microcephaly, as well as the general form, is irreversible, and no treatment for either has been established.

Case example of microcephaly with idiocy—A boy of five years, diagnosed as microcephalic, was found to have a history suggesting maternal biological inadequacy. The mother had four pregnancies, the first of which had produced a hydroencephalocele who died in a few hours, the second a miscarriage, the third a normal girl, and the fourth the patient. The mother was not luetic nor alcoholic, nor had she any illnesses more serious than an attack of bronchitis during the fourth pregnancy, which went to full term. In the entire 9 months she gained only 6 pounds. The labor was slow, intermitting for 24 hours, the cervix rigid and amniotic fluid excessive. Outlet forceps were used for delivery. The infant at birth weighed 7 pounds, 1½ ounces, had tight cranial sutures

and very small, almost closed fontanelles, and was unable to nurse. Teething was normal, but he was puny and delicate and did not cry normally. When examined at age 5 he weighed 20 pounds, walked with support, slept very poorly, and had temper tantrums and no bowel or bladder training. Formal psychometric studies were impossible, but the general estimate of his mental age was from 9 to 11 months.

D. *Macrocephaly* is enlargement of the cranium due to enlargement or hypertrophy of the brain, and is accompanied by various degrees of mental defect. The brain is not only excessively large, but is also thickened, with hypertrophy of the cortex and the subcortical white matter. Most cases show profound mental defect, either low grade imbecility or idiocy, with convulsions, defective vision and small, frail skeletal and muscular development. Death usually occurs before adult life as a result of severe convulsions or intercurrent infections. The family history of macrocephalics shows an unusual saturation with neuropathic disorders.

E. *Hydrocephaly* is the accumulation of an abnormal amount of cerebrospinal fluid within the cranium, which may in some cases become greatly enlarged. The fluid exerts increased pressure either against the surface of the brain (external hydrocephalus) from its accumulation in the subarachnoid space, or from within the ventricles (internal hydrocephalus) due to damming up by defects closing the communicating foramina or to defective formation of the subarachnoid spaces and cisternae. Both conditions result in atrophy of the brain and mental deficiency, and may be associated with a variety of defects of the nervous system. The only encouraging report to be found in decades of writing relative to hydrocephaly is that of the operative treatment initiated by Dandy and Putnam in 1938. This technique is to remove the glomus of the choroid plexus of both lateral ventricles. The operation is more likely to be effective in older infants with slowly progressing hydrocephaly, since the rapid development of this disease endangers brain substance and early treatment is necessary to prevent mental retardation. Operation after the disease is established is seldom rewarding. The results of endoscopic cauterization of the choroid plexus through trephine openings over the occipital lobes have been reported recently by Scarff. Of 10 cases successfully operated upon four progressed with normal mental development, three were moderately retarded, two became definitely defective and one died of intercurrent infection.

F. *Craniostenosis* is defective development of the cranium caused by premature or faulty synostosis of the cranial bones, due probably to defects in the germ plasm. In this disorder premature fusion of the cranial bones prevents the skull from expanding normally to accommodate the growing brain and causes increased intracranial pressure, atrophy of the cortex and a severe degree of mental deficiency. There are many possible malformations, but those most commonly recognized are oxycephaly or "tower skull," acrobrachycephaly and scaphocephaly. Ford states that if the diagnosis is made before optic atrophy and severe brain destruction have occurred, surgical measures to relieve intracranial pressure are indicated. In true oxycephaly the only feasible operation is believed to be bilateral subtemporal decompression. Woodhall recommends morcellation of the skull to relieve the threat of mental defect resulting from oxycephaly. The purpose of the operation of morcellation is to increase the intracranial space symmetrically, to preserve the bony covering of the brain and to reduce the exophthalmus and preserve vision. The operation relieves the increased intracranial pressure, obliterates the "tower skull" deformity and in some instances some gain in mental functions can be expected. In other types of craniostenosis, in which only one suture is synostosed, Ford believes it possible to relieve the condition by merely channeling the bone along the suture line.

C. *Cranium bifidum* or incomplete closure of the cranial sutures, is associated with cerebral dysplasia and mental deficiency. The defect usually occurs in the sagittal plane and most frequently is either frontal or occipital. The severest disorder resulting from this condition is meningoencephalocele, in which a hernia is formed containing portions of the brain and ventricular system covered with meninges and skin. In cranium bifidum occultum no herniation is present but according to Ford there may be cerebral defects, convulsions, blindness, and numerous other complicating features. Penfield states that

simple meningoceles may be excised successfully but that no amelioration of mental condition has resulted.

H. *Neurofibromatosis*, or Recklinghausen's Disease, in which cerebral development is interfered with by the presence of neurogenic tumors, is associated with localized signs of intracranial tumor. In a study of 8 cases Rosendal found acoustic neuroma, optic glioma, tumors of the brain stem and cerebrum, multiple meningiomas and diffuse arachnoiditis. Seventy-five per cent of cases show advanced mental deficiency, 15 per cent show character defects, and others display various degrees of asocial behavior, delinquency, tendency to steal, etc. The causation is unknown and surgical treatment is not advisable except in cases where larger tumors threaten vital functions.

I. *Tuberous sclerosis*, or the presence throughout the body of small sclerotic masses, is a disease of rare occurrence and unknown etiology. It is believed by Marchand et al. to be a developmental defect of the nervous system which begins in the embryonic stage and progresses through the life of the victim. They cite a case of a malignant form which ended fatally at age 6. The mental picture was that of idiocy with violent aggressive behavior and motor agitation. Histologic studies of the sclerotic areas showed them to be atypical neurinomas and ganglioneuromas. Alexander and Myerson studied the cell mineral content of nerve tissue in cases of tuberous sclerosis and found hypermineralization of the cytoplasm. They conclude that the disease resembles the neoplastic disorders rather than the degenerative types.

J. *Cerebral dysplasia of prematurity* favors the occurrence of cerebral birth trauma, as has been demonstrated by post mortem examinations of premature infants showing that 90 per cent have cerebral lesions. Intracranial hemorrhage was found by Browne to occur 16 times more frequently in premature than in full term infants, and by Levinson to occur 42 times more frequently. There is little statistical knowledge of the causation of prematurity. It is demonstrably favored by maternal trauma, endocrine deficiency and toxic states, and probably by shock and nutritional deficiency. According to Alpers it is often caused by some systemic disease of the mother, or by other factors which favor also the occurrence of cerebral birth trauma. The premature brain is poorly developed, is softer and has a higher water content than normal, and hence is more vulnerable to the forces and strains incident to birth. A further factor in the etiology of mental and personality defects is the cellular immaturity in the premature brain. The generalized character of the defects would point to the existence of an encephalopathy resulting from the diffuse cellular lack in the cerebrum. In 122 cases of mental defectives Rosanoff and Inman Kane found a definite history of prematurity, which had occurred in 21.3 per cent. In cases of prematurity, as in those of encephalitis, cerebral diplegia and many other cerebral injuries, the encephalopathy may seriously affect the intellectual functions, or may do no more than create defects of emotional adaptability or personality.

K. *Other forms* of developmental structural pathology which cause serious mental defect are degeneration of the central gray matter (Alpers), chronic infantile cerebral sclerosis (Pelizaeus-Merzbacher), subacute juvenile cerebral sclerosis (Scholz), acute infantile cerebral sclerosis (Krabbe), neuromyelitis optica, disseminated encephalomalacia with cavity formation, and other types less completely described in the literature.

V. *Mental Defect Due to Endocrine Disorders*

In many ways the study of endocrine defects in relation to mental retardation is the most encouraging branch of the whole problem of mental defect. Not only is the therapeutic result more satisfactory but with better understanding of the maternal endocrine factors in the etiology of infantile endocrinopathies the likelihood of prevention increases very materially. The data on incidence of endocrine disturbances associated with mental deficiency are extremely inconsistent, depending on the investigator's point of view as to whether the endocrine defect is considered to be the sole etiology or merely a complicating or possible causative or aggravating factor. Studies of large numbers of institutionalized defectives, however, generally agree that the occurrence of endocrine pathology averages about 20 to 25 per cent. Kimball and Marinus made an analysis of 3548 institutionalized cases of mental deficiency and found 712 presenting unmis-

takable evidence of severe glandular disturbance. Of this number 281 showed a direct etiological relationship between the mental defect and severe congenital hypothyroidism. The remainder displayed pituitary disorders, combined thyroid and pituitary disease, adrenal cortical disorders, gonadal insufficiencies or maldevelopment, etc. There have been numerous contributions to the literature showing not only the relation of endocrine disturbances to mental retardation but their relation to behavior disorders, delinquency and personal maladjustment. In our juvenile court survey 15 per cent of offenders suffered from endocrine defects which were directly or indirectly causative. In a study made by Rowe of 650 cases under 17 years of age, all presenting various combinations of behavior disorders and intellectual retardations, 68 were found in whom endocrine disturbances could be considered causative. A survey made by Molitch of 500 boys committed to a state correctional institution showed 25 per cent to be suffering from endocrine disturbances. The large majority of these cases were mentally retarded. In general, thyroid and thyropituitary disorders have the most profound intellectual and emotional defects. Englebach states that all untreated congenital and infantile hypothyroid conditions result in definite feeblemindedness, 50 per cent of thyropituitary dysfunctions are mentally retarded and approximately 10 per cent of pituitary dystrophies have some intellectual impairment although the most characteristic disturbances are in the emotional sphere.

In a study of the neuropsychiatric aspects of adolescent endocrinopathies 15.5 per cent of 177 cases were found to have mental retardation. Of the mentally retarded cases adequately treated by endocrine substances 44 per cent showed improved mental capacity. Twenty-seven per cent of 177 cases of adolescent endocrine disorders were found to be emotionally unstable, excitable, erratic, depressed, irritable and in other ways suffering obvious emotional imbalance. Sixty per cent of cases adequately treated by endocrine therapy showed marked improvement. Seven per cent of the 177 had epileptic states of varying severity, accompanied by the usual personality deviations (Englebach).

In adolescence the mental retardation associated with endocrinopathies is a more serious problem than in the infantile or juvenile age groups. Under the general physiological and psychological stress incident to puberty many latent or heretofore symptomless glandular disturbances may develop into severe incapacitating illnesses. It is an error to assume that an unrecognized pre-adolescent endocrine defect having become clinically prominent during puberty will pass with the completion of pubertal changes or be "outgrown." At adolescence it is already late to expect the optimum treatment results although every effort should be made to correct the defect. Hypothyroidism presents at this age its characteristic symptoms of mental sluggishness, physical inertia, excessive weight, typical body contour and intellectual dullness with usually an added symptom of depression with feelings of inadequacy and failure. The cases of pituitary dystrophy usually begin in adolescence to display greater instability and more erratic behavior with the dependent, immature aspects of the personality becoming more obvious as their more normal school mates and friends achieve maturity and character. In addition the pubertal changes are retarded and this adds another psychic factor of sexual inferiority which in those of limited intelligence may lead to aggressive, destructive compensations.

A. *Hypothyroidism*—The importance of heredity in the etiology of infantile endocrinopathies is an established fact. Although in some cases of congenital deficiency the thyroid gland has been damaged by maternal infections or by severe toxemias of pregnancy, the large majority result directly from maternal endocrine imbalance. Thyroid deficiency in the pregnant mother is not difficult to diagnose since a carefully taken history will reveal such positive evidences as menstrual irregularities, delayed, scanty periods, the physical signs of characteristic adiposity and myxedematous subcutaneous tissues, and lack of normal rise of metabolism (to plus 10 or 15 per cent) during the third to sixth months of pregnancy. According to Englebach, it is possible to minimize the chances of intellectual injury to the offspring by correction of the maternal glandular deficiency during the pregnancy. Since there is proof of the inherited inactivity of the thyroid due to decreased supply of thyroxin to the fetus adequate thyroid medication of the mother is of

paramount importance.

In congenital hypothyroidism or cretinism the retardation of mental and physical growth is obvious and is present from birth. Early recognition is of the utmost importance if serious psychological and somatic defects are to be prevented. However, in more than 2000 cases of endocrine disturbance Englebach found only 3 per cent recognized by the parents or family physician in early infancy while from a carefully taken developmental history it was obvious that in 30 to 40 per cent the endocrine defect had been present in the early months of life. Infantile hypothyroidism most commonly results from hereditary or congenital endocrine defects but such factors as nutritional deficiencies in infancy, early infectious diseases and iodine deficiency are of considerable etiologic importance. Of the infectious diseases chicken pox, measles and scarlet fever appear to be the most hazardous as far as thyroid damage is concerned. Diagnosis can be established by the definite history of retardation in physical growth and mental development and by x-rays of the knee and ankle which show delayed osseous development. It is rare for severe cases of infantile hypothyroidism to achieve a mental age of more than eight years even under thyroid treatment, which fact again stresses the importance of recognition and treatment in the earliest months of life.

In juvenile hypothyroidism the facts most characteristically found in the history are, in order of their frequency, arrested mental development, retarded physical growth, a history of myxedema in the mother. X-ray of the carpal bones will show delays of ossification often as great as two to three years, and together with delayed body growth is a valuable criterion in both diagnosis and control of treatment. Therapeutic test with administration of thyroid extract up to the child's tolerance will of course further clinch the diagnosis. Rapid improvement in both physical and mental growth can be anticipated. Beneficial results are due not only to acceleration in physical growth, but to stimulation to normal activity, more normal appearance and improved social adjustment. It is doubtful that thyroid therapy directly affects the basic intellectual capacity to any substantial degree.

For a study of the effect of thyroid medication on the brain metabolism of cretine, Himwich, Daly, Fazekas and Herrlich chose 11 cretins ranging in age from 9 to 31 years. They were observed with regard to both the intrinsic cellular oxidation and the integrative action on the organism. A thorough study indicated that the increase in brain metabolism averaged 32 per cent as the result of thyroid administration. The electroencephalograms disclosed a rise of the energy levels in certain frequency ranges. From the standpoint of increased mental capacity the improvement was either absent or of questionable significance. The greatest mental change was that showing acceleration of psychological activity made possible by the increased energy expenditures stimulated by thyroid medication. An increase in mental age of cretins one to five years old treated with thyroid extract is reported by Bronstein and Brown. They believe that from the prognostic standpoint a cretin, if treated early in life, is not unpromising material.

B. *Pituitary dysfunction*—Pituitary dystrophies have comparatively little intellectual deficiency, estimated at 10 per cent of all cases, but the emotional infantilism characteristic of the disorder can render the individual unadaptable to a marked degree. The emotional instability, peevishness, immaturity and antisocial impulses typical of the majority of children suffering from Frohlich's syndrome are a constitutional stamp conditioned primarily by the endocrine abnormality and only to a minor degree by environmental and compensatory factors. In the etiology of Frohlich's syndrome hereditary factors are of importance, there being a strong evidence of thyroid and pituitary disorders in the family history in a majority of cases. There are, however, many instances in which toxic, infectious or traumatic damage appears to play a direct causative role, and in such cases it is impossible to state positively that the endocrine factor is primary in the mental retardation since the trauma may have been sufficient to do organic brain injury of considerable severity. A behavior disorder reminiscent of post-encephalitic conduct is typical of the more severe deviations associated with Frohlich's syndrome. The Lawrence-Moon-Biedl syndrome is a relative rarity although cases are reported in the literature at the rate of several per year. The outstanding

features are symmetrical polydactylism, syndactylia, obesity, retenitis pigmentosa, squint, nystagmus and mental deficiency. Postmortem examination of the pituitary in a case of this kind was made by Griffiths who found the presence of a large proportion of basophile cells in the anterior lobe of the pituitary. Wolff found evidence of hypothalamic as well as hypophyseal pathology in the disturbances of water metabolism which occur in the syndrome. Water balance was restored on administration of anterior pituitary substance. Occasionally Frohlich's syndrome and Cushing's Disease are associated with tumor of the pituitary gland. If such is the case, symptoms and findings of optic chiasm compression with bitemporal visual disturbances should be present. In rare cases enlargement of the gland may produce compression of the third ventricle with symptoms of increased intraventricular pressure (hydrocephalus). When such a combination of symptoms is present the indications are for surgical intervention.

The following case illustrates the difficulty of differentiating between traumatic causation and hereditary endocrine causation of mental deficiency.

Case example of pituitary deficiency, trauma and mental retardation—A girl of 10 years, 4 months, was diagnosed as a case of thyroid-pituitary dystrophy. The first born, her birth had been 6½ weeks premature, following a normal nine-hour labor and normal, spontaneous delivery. The weight at birth was 6½ pounds and a fever was present for the first three days of life. As an infant she never cried normally, was excessively obese, vomited frequently and was retarded in learning to walk and talk. At 1½ years of age she fell 6 feet through a trap door into the cellar, was unconscious for one hour and afterwards was harder to manage, beating her head on the wall in tantrums, etc. At 4 years, 9 months, she had appendicitis complicated by pneumonia, with a temperature of 105°, followed by pyelitis. At the time of examination at age 10 her behavior was marked by infantilism and excessive friendliness with indiscriminate caressing and embracing of persons in her company. She played with children smaller than herself, sucked her thumb and fingers, was subject to headaches, had a bad scholastic record and practiced masturbation. She tore up and ate paper, slept poorly, and screamed and struck when angry. She was restless, inattentive and defiant, easily offended and jealous, with slouching attitudes, bad table manners and silly talk. Her height was 4 feet, 9¾ inches, weight 111 pounds, and the physical contour typical of Frohlich's syndrome. The pituitary fossa was 7x6 mm. Psychometric findings were as follows:

Stanford-Binet:	
Mental Age	8 years, 2 months
I. Q.	80
Basal Mental Age	6 years
Kohs Block Design:	
Mental Age	6 years
I. Q.	58
Educational Age	6 years, 10 months
Social Age	9 years, 2 months

Endocrine therapy was prescribed in the form of daily doses of 1 grain thyroid extract, 4½ grains whole pituitary and 9 grains extract of anterior pituitary lobe. After five years of this treatment the patient was examined again at age 16. Her height was 5 feet, 6 inches, weight 141 pounds, and her behavior was more normal in every respect. Her I. Q. had not materially changed.

C. *Adrenal cortical hyperplasia* may result in a serious condition both from the psychiatric point of view and as a threat to the physical well being and survival of the patient. There are various terms used in the literature, the most common of which are virilism, macrogenitosmia, pubertus praecox, hypergenitalism, hypergonadism, etc. The primary feature of the syndrome is the marked acceleration of sexual maturity, the child reaching a state of adult sexual development often as early as the fourth year. Other characteristics of the disorder are premature osseous maturity readily shown on x-ray examination, retarded growth in height, arterial hypertension, marked hirsutism, inordinate appetite, polydypsia and pigmentation of the skin. The mental retardation often reaches as severe proportions as that of cretinism, but in addition to the intellectual defect there is a fairly typical behavior disorder, the chief feature of which are emotional instability, irritability, lack of adequate inhibitory control, sexual aggressiveness, restlessness, de-

structiveness and general inadaptability. The mortality rate is relatively high, death usually occurring before the 20th year.

Case example of adrenal cortical hyperplasia—A boy of seven with an endocrine disorder presented a moderately severe case of adrenal cortical hypergonadism. His behavior was marked by a consistently antisocial attitude with truancy, stealing, vandalism and aggressive sexual acts undoubtedly motivated directly by his sexual precocity. His mental age was 5 years, 3 months. There were present the typical somatic changes of advanced ossification (near adult), arterial hypertension and hirsutism. The voice was gruff and coarse, the genitalia were adult and live spermatozoa were demonstrable in the seminal fluid.

D. *Mental defect due to combined types of endocrine disorders*—Mental defect is often associated with combined types of endocrine disorders such as juvenile osteochondral hypothyroidism, the so-called pituitary myxoedema, infantilism (Rothmund-Werner type), the so-called idiotia thymica, and Albright's Disease, which is a fibrous dysplasia of bones with endocrine disorders and cutaneous pigmentation with occasional severe involvement of the skull bones. Gargoylism has many combined endocrine features although it has never been classified as a distinct endocrine entity. The endocrine features are the bone malformations with the cranial defects, changes in vertebral bodies, deformity of the spine and limbs. In addition there are multiple opacities of the cornea with impairment of vision and enlargement of the liver and spleen is usually present also.

VI. *Mental Defect Due to Hereditary or as Yet Unknown Influences*

A. *Amaurotic family idiocy*—Little progress has been made in the explanation of the nature of this disorder since the original contributions of Tay, Sachs, Bielschowsky, Hassin, Pick, Jansky, Vogts, Kufs, Jervis, Spielmeyer, Globus, Meyer and others. The division of the condition into the four general types listed in the outline was suggested by Rothstein and Welt in 1941. The infantile or Tay-Sachs form appears related to a disturbance of lipoid metabolism, and is always characterized by blindness, profound dementia and an acute course ending in death at about the age of 2 or 2½ years. Alexander and Myerson studied the cell minerals in the cerebral tissues of cases of the infantile type by means of microincineration and spectroscopy. The ganglion cell content was characterized by reduced calcium and phosphorous and increased lipoid material; also by demineralization, which would place the condition among the degenerative diseases, rather than among the neoplastic or inflammatory types. Six cases of the juvenile type of amaurotic idiocy (Vogt's) were reported by Jervis in 1941. In all these cases the first symptoms appeared in apparently normal children between 5 and 6 years of age, and consisted of progressive blindness due to primary optic atrophy. Mental deterioration appeared shortly after the visual symptoms and 2 cases became psychotic. Generalized convulsions followed at ages 9 to 11 and spastic flexion attitudes developed between ages 13 and 14. Death occurred in all cases between 14 and 16 years of age.

B. *Phenylpyruvic oligophrenia* is an inborn metabolic error characterized by mental deficiency and excretion of phenylpyruvic acid in the urine. The blood of 16 patients with this condition was studied by Jervis et al. who found a phenylalanine blood content varying between 15 and 40 mgms. per cent. They believe that the essential biochemical characteristic of the disease is inability to dispose of the phenylalanine at the normal rate rather than failure to break down phenylpyruvic acid. The disease itself is not fatal.

C. *Neuromuscular disorders* do not in themselves necessarily produce mental deficiency but are often found associated with it. Among them may be cited spinal muscular atrophies caused by degeneration of the motor cells of the spinal cord, with muscular wasting and paralysis. The Werdnig-Hoffman or infantile type is familial, with onset between the 6th and 12th months of life, having symptoms of weakness and flaccidity of the legs and later of the entire trunk, followed by contractures and deformities, and ending in death after 4 or 5 years. Amyotonia congenita is apparent at birth, as is also atonic congenital diplegia. In the three latter types the onset of disease occurs so early in life and is so completely disabling that they are invariably associated with mental deficiency. Whether this is due to cerebral destruction by the same process as that which causes the

neuromuscular defect, or whether it is due to inhibition of mental growth by grave physical disability has not been ascertained. Other types are myasthenia gravis and myatonia congenita (Oppenheim). In the latter disorder the findings in the nervous system and muscles would seem to indicate developmental retardation and malformation. The muscle changes apparently parallel the central nervous system alterations. Histologic studies of the nervous system indicate also that certain tracts are not myelinated as late as the 3rd month of life and that the myelination of cerebral white matter is also incomplete. Recent steps in the treatment of neuromuscular disorders are somewhat encouraging. The benefits of prostigmine in myasthenia gravis are emphasized by Eaton, who also finds growing evidence of the causative relationship between thymic disorders and myasthenia. Weinberg has reported on the value of adrenal cortical hormone in the treatment of myotonia congenita.

D. *Friedreich's ataxia* is a degenerative disorder involving chiefly the fiber tracts of the spinal cord, the cerebellar apparatus and the motor nuclei of the brain-stem and spinal cord. The first symptoms appear in childhood and include retardation in walking and writing, clumsiness and a tendency to stagger and fall and spill food. Recognition of these early signs is important since prompt diagnosis is occasionally overlooked because the early symptomatology is attributed to natural slowness or clumsiness in the child. As the disease progresses there are marked incoordination, optic atrophy, deformities, helplessness, bedridden invalidism and dementia. Patients may live for many years.

E. *Schilder's Disease* (Schilder-Fox) or encephalitis periaxialis diffusa, is a degenerative disorder associated with massive focal lesions in the cerebral white matter, first involving the myelin sheath and later causing softening of the cerebral tissue. It is a similar condition to neuromyelitis optica, which is characterized by focal degeneration of both brain and spinal cord, involving gray as well as white matter and sometimes causing complete softening of the entire brain and cord. The early symptoms of Schilder's Disease, usually appearing in childhood, are hemianopsia progressing to cortical blindness, followed by motor disturbances such as hemiplegias, aphasia, apraxia, ataxia and convulsions and by cortical deafness. There is usually a rapid advance to dementia, with noisiness, screaming, apathy, coma and increased intracranial pressure with headache. Death usually occurs within one to two years. The early diagnosis of Schilder's Disease may be a matter of considerable difficulty and the course is not always rapid. The following case, having a variety of diagnoses and an unusually prolonged course, illustrates some of the interesting problems involved.

Case example of Schilder's Disease—A boy of 12 was admitted to the Pennsylvania Hospital, Department for Mental and Nervous Diseases, in 1930. His birth and early development were normal and he suffered only the usual childhood diseases. As a child he was alert, bright, active and eager to learn. At the age of 7 he fell off his pony and was dragged for some distance. Immediately following the accident he was somewhat bewildered but was not unconscious. The following day he was irritable and drowsy and then for three weeks was semi-stuporous, and had major convulsive seizures but no fever. There were no signs of intracranial bleeding. Neurological signs were minimal, there being only variable diminution or absence of deep reflexes and inconstant Babinski's sign bilaterally. After five weeks in the hospital he was mentally confused, walked with an ataxic gait, and was aphasic and apraxic. Five months later he was still aphasic, was irritable, had fits of temper and petit mal spells and exhibited an odd "tossing of the head and body." Six months later he developed generalized convulsions, had lost all ability to feed and clothe himself, was incontinent and was suffering progressive impairment of vision. He appeared to be deaf but had not sufficient mental capacity to make accurate estimates of auditory function possible. In 1929 at the age of 11 years an operation on the brain was undertaken with the thought that there might be some "silent" hematoma with scarring, etc. A large left fronto-parietal flap was made and a mass of varicose veins was found centering about the anterior end of the Sylvian fissure. These veins were tied off. In 1930 examination showed bilateral pyramidal tract involvement, muscular weakness, reeling gait, aphasia, impairment of vision and hearing and muscular twitching of the body. Generalized convul-

sions occurred as often as 12 to 20 times daily. Some improvement followed ketogenic diet, fluid limitation and phenobarbital therapy. The general deterioration continued, however, and the patient died in 1933 at the age of 15 years.

Autopsy report: There is marked thickening of the dura and pia arachnoid with dense adhesions throughout but especially marked at the site of operation.

Grossly the brain is small, with symmetrical subcortical degeneration more marked on the right, and involving the precentral, postcentral and parietal areas, and the right frontal region. There is dilatation of the posterior ventricular horn. There is some convolutional atrophy and microgyria.

Histological examination by Dr. B. J. Alpers: The outstanding process appears to be the marked gliosis of the white matter. In many parts the white substance is filled with glia which are chiefly hyperplastic astrocytes but with some oligoglia among them. This gliosis appears not to be strictly confined to the white matter and spreads also into the underlying adjacent cortical layers, sometimes into the entire cortex. The population of the white matter is so dense that it takes a much darker stain than the cortex. The Weigert section shows a definite demyelination of the white matter. This involves all the fibres in the gyrus including the arcuate fibres. Sometimes the demyelination involves two or more adjacent gyri. The cortex is also demyelinated. The process is a degenerative subcortical encephalopathy characterized largely by gliosis without hematogenous elements. Diagnosis: Degenerative subcortical encephalopathy (Schilder's Disease).

F. *Huntington's chorea* and similar conditions such as dystonia musculorum deformans and other athetoid states have been found to involve selective degeneration of basal ganglia and forebrain, with consequent intellectual impairment. The onset is usually thought to occur between 30 and 40 years of age, but many cases have been known to occur in childhood as early as age 4. It is noteworthy that mental symptoms are occasionally the earliest to appear, such as irritability, emotional instability, excitability, destructiveness, antisocial acts, delusions, etc. In other cases the earliest symptoms may be choreiform movements, grimaces, exaggerated voluntary movements and incoordination, followed by bulbar disorders and loss of speech. The disorder is progressive and ends in death 10 or 12 years after onset.

G. *Wilson's Disease* (hepato-lenticular) is a degenerative process affecting the brain, especially the lenticular nuclei, and involving progressive mental enfeeblement which in some cases reaches the point of severe dementia. The disorder shows a close resemblance to the Parkinsonian syndrome, with athetoid movements and loss of the power of speech. Onset is usually in the first decade of life. Recent investigators believe that disturbance of liver function is the primary cause of this condition. Neurological symptoms appear after the cirrhosis is advanced, and death occurs 2 or 3 years later.

H. *Idiopathic epilepsy*—The part played by epilepsy in the etiology of mental defect is difficult to evaluate. A new phase in the study of this disease began with the adoption of the electroencephalograph as a diagnostic instrument, since by its use it became possible to differentiate between idiopathic epilepsy and other convulsive or petit mal like conditions presenting a similar clinical picture but showing variations from the typical epileptic cortical dysrhythmia. Since it is convenient to treat the subject of epilepsy under a single heading, both idiopathic and other forms of this disorder will be discussed here. Only idiopathic or essential epilepsy, however, will be considered as an etiological factor, because other convulsive disorders are accompaniments more often than causes of mental defect, and any attempt to distinguish between these two relationships would in the present state of medical knowledge result only in confusion.

Conditions frequently accompanying mental defect, and presenting the clinical syndrome of epilepsy, caused by trauma, infections, toxic states, endocrine or nutritional deficiencies, etc., can be treated by various methods appropriate to their etiology. In cases of cerebral trauma, the most frequent cause of severe accidental convulsive states, surgical excision of scar tissue has been practiced with marked success by Penfield, who uses electroencephalographic tracings both to differentiate traumatic convulsive states from idiopathic epilepsy, and for the localization of irritative focal lesions. It is believed by Pen-

field and Keith that the percentage of cases of traumatic epilepsy due to birth injury is considerably higher than the accepted figure of 3 per cent. They state that epileptogenic lesions dating from injury to the brain or meninges at birth or in infancy include: (1) areas of focal atrophy; (2) meningocerebral scar caused by depressed skull fracture leaving dense adhesions with anastamosing blood vessels, connective tissue and ganglia; (3) infantile meningitis with dense vascularized subdural adhesions, leptomeningeal thickenings and underlying cortical injury by circulatory disturbances; and, (4) focal cerebral thrombosis secondary to arterial occlusion. These lesions, which may produce various degrees of mental defect, are often amenable to neuro-surgery. It is stated by Fay that the incidence of chronic subarachnoid fibrosis, sometimes accompanied by mental defect, is high among clinical epileptics and is for the most part attributable to birth injuries and subarachnoid bleeding.

Results attained in traumatic cases and in the amelioration of other epileptiform disorders by detoxification and correction of nutritional, vitamin and endocrine deficiencies, indicate that accompanying mental defect or retardation is in such cases usually a result of the underlying condition rather than of the convulsive states engendered by it; although it is believed by Thom that convulsions per se may cause cerebral damage which affects normal brain development and results in mental deficiency.

The importance of idiopathic epilepsy in the causation of serious mental defect cannot yet be determined statistically because electroencephalography has not yet come into full use as a diagnostic criterion and data based on its findings are not plentiful.

In a study of the relation between convulsions of early life and chronic convulsive disorders in cases of mental defect, Thom states that idiopathic convulsions occurring in early childhood are more likely to merge directly into chronic convulsive disorders than are convulsions of other types except those associated with manifest brain damage. Petit mal attacks occurring during the first two years of life are likely to merge into chronic convulsive disorders or become associated with mental retardation. A study is reported by Brown and Solomon of 20 boys committed to a state training school as delinquents, most of whom were intellectually retarded. Seventeen of the 20 yielded abnormal electroencephalographic tracings, the records indicating petit mal. In a study by Lennox of 1900 idiopathic epileptics, 64 per cent were found to be intellectually normal and only 14 per cent seriously deteriorated, in spite of the fact that the average patient had experienced 2000 seizures during a period of eight years. He urges that mental deterioration in epileptics be considered as arising from one or more of five possible causes: (1) constitutional tendency or heredity; (2) brain injury which antedated the onset of the seizures; (3) overmedication with sedative drugs; (4) environmental, including psychological and social, stresses; (5) the condition underlying cerebral dysrhythmia. Heredity undoubtedly plays a part in the impairment of mentality of certain patients subject to seizures. So also does the constant depressant effect of sedative drugs given to control the seizures, as was shown by the heavy witted, sluggish state commonly seen in epileptics in the past, which has given place to improved mood and mental acuity following the use of the newer non-sedative therapeutic management. Environmental interference with mental efficiency can be seen in the emotional changes resulting from the psychological hazards inherent in the constant state of insecurity resulting from the fear of attacks.

Mental and temperamental peculiarities caused by the organic condition underlying cerebral dysrhythmias of epileptic type have been studied by Palmer and Hughes, who found by the use of the electroencephalograph that such personality defects existed in a large number of cases who had epileptic cortical dysrhythmias but did not exhibit the accepted grand mal or petit mal diagnostic features of the disease. They found that such subclinical epileptics were frequently classed as neurotics or behavior problems, and exhibited a fairly consistent pattern of mental and emotional abnormality. The chief characteristic features of this abnormality were found to be faulty inhibitory control, moodiness, irritability, impulsiveness, intolerance of frustration and tangential, inconsecutive ideation, together with blunting of perceptions and of normal emotional responses, the whole adding up to a picture of erratic behavior and lack of

responsibility and common sense. These authors conclude that there is dysrhythmic ideation, emotion and conduct in the epileptic personality, whether the epilepsy is of clinical or subclinical degree, and cite examples of abnormal behavior in children. A child who cut off a cat's tail with a pair of scissors, another who bit a playmate's toe so savagely that it required surgical sutures, another of exemplary bringing up who threw a brick through a large church window, and another who built a bonfire on the dining-room rug, were all found to have epileptic dysrhythmias. It is estimated that while dysrhythmias are relatively common, only 5 per cent of persons with dysrhythmias have actual convulsive or petit mal epilepsy. The remaining 95 per cent, who can be accurately diagnosed only by means of electroencephalographic tracings, are likely in their early years to fall into the class of delinquents or scholastic laggards.

The management of epilepsy by means of the synthetic anticonvulsant drug dilantin sodium and the ketogenic diet has removed at least one possible cause of mental deterioration in the patients, since this drug produces no hypnotic dulling effects. It is moreover not only effective in controlling the seizures, but causes the cortical rhythms to return to normal or near normal. Price, Waelsch and Putnam have recently employed dl-glumatic acid hydrochloride in the treatment of petit mal and psychomotor seizures with considerable success. It is used alone or in conjunction with dilantin sodium, and is administered orally in capsule form. Increased mental and physical alertness have been the most noteworthy side effects and the relief from the petit mal and psychomotor seizures has been remarkable. The ketogenic diet also appears to have some basic therapeutic value as well as anticonvulsant effects. It is reported by Wilkins that a number of patients remained free from attacks during the entire course of a ketogenic regime and did not have a recurrence of seizures on return to a normal diet. Helmholtz and Goldstein report that there have been no attacks or convulsions for six years in 90 per cent of their cases on ketogenic diet, and they believe that only 5 to 10 per cent will have a return of seizures after the restoration of a normal diet. It is believed by Palmer and Hughes that the management of epilepsy today demands that the aim of the treatment be not solely the elimination of the attacks but the preservation of intellectual powers and the restoration of the cortical rhythms as nearly as possible to normal.

I. *Mongolism*—The role of endocrine pathology in the etiology of mongolism has been the subject of recent studies, which indicate that a definite pattern of thyroid and pituitary abnormality is a characteristic feature of the disease. A discrepancy between the histologic picture of the thyroid gland and the age of the child was found by Benda. The gland changes resembled those observed in various forms of goiter and also those produced experimentally by administration of the thyrotropic principle of the anterior lobe of the pituitary. They suggested a temporary stage of either hyperthyroidism or thyroid dysfunction. Studies of the pituitary by the same investigator have shown a lack of basophiles during the first years of life and later a marked deficiency of basophiles in the anterior pituitary, with replacement by an increased number of acidophiles. The anterior lobe of the pituitary is known to influence both the architectural variations of the skull and the growth of the central nervous system and consequent mental development. Its fundamental importance in this form of idiocy is indicated by Benda's study demonstrating a slowing down process in the growth of the skull with retention of fetal characteristics and a general hypoplastic growth condition. It was found, however, by Werner et al. that administration of thyroid extract and growth factor of the anterior pituitary lobe failed to affect the growth or mental state of mongols. The origin of this disorder is attributed by Rosanoff and Handy partially to factors operating early in the embryonic period of development. Its occurrence in relation to age of the mother makes it appear to have some connection with ovarian pathology or ovarian exhaustion. Lower than normal cerebral metabolism was found in mongols by Himwich and Fazekas. There was less oxygen consumption, reduced sugar uptake and the circulation time was rapid. Electroencephalographic tracings of mongols show no variation from normal, conforming with the general picture of a pathology without cerebral focal points and without abrupt neurological variations. Alexander and Myerson found that the cere-

bral tissue of mongols did not differ materially from normal in mineral content as determined by microincineration and spectroscopy.

J. *Other types* of hereditary or unknown possible causes of mental defect are Niemann-Pick's Disease, hereditary amyotrophic lateral sclerosis, hereditary bulbar palsy, familial cerebral calcification (Penfield), Leber's optic atrophy and a new disorder recently described by Jervis, a curious form of severe idiocy occurring in 6 cases among nine siblings of one family. The syndrome is characterized by retarded body growth resembling gargoylism in some ways, with low hair line, bushy brows, large depressed bridge of the nose, protruding lips, well developed jaw and peculiar changes in the bones of the skull. The cranial bones show thickening, widely separated tables, spongy diploe, coarse trabeculae and increased density on x-ray. The brain shows general atrophy. Jervis regards the condition as a form of lipidosis.

VII. *Mental Defect Due to Indirect or Environmental Causes*

A. *Psychological disorganization*—It has been abundantly demonstrated that impairment of intellect can be produced not only by injuries arising directly from tissue destruction or chemical change, but also by those influences which interfere with the acquisitive power of the individual during the developmental years. Although such cases are comparatively rare, it is well known that devastating injury from trauma or infections, not primarily involving the central nervous system but sufficient to disrupt the personality integration of the child, may so seriously impair the acquisitive capacity as to result in failure of development. In the same way some of the lesser physical defects, such as impairment of vision or hearing, may in some instances diminish the competitive spirit and rob the child of the desire to achieve, so that accumulation of information is blocked. These are some of the ways in which it is possible for organic disorders indirectly to interfere with intellectual growth, and they are of course extremely variable and are modified by environmental influences.

Summary

1) A revised grouping of forms of mental defect based on etiology is offered as a flexible working basis rather than as an attempt to introduce into the literature a complete detailed classification. It is expected that as newer knowledge sheds more light on causative factors a more precise classification can be drawn up. The physiological attack on the problem of mental defect has recently expanded our knowledge in some fields and has made necessary the transfer of many pathological entities from the hereditary or congenital (primary) group to the group of biochemical or physiological (secondary) disorders. In the progress of these studies a number of remediable causative factors have been recognized and in some fields there are encouraging therapeutic possibilities.

2) In mental defects due to hereditary or as yet unknown causes, numerically the greatest etiological category, outstanding progress has been made in the diagnosis and management of epilepsy, particularly by means of the electroencephalograph and the development of a non-sedative, anti-convulsive drug. A certain foundation has also been laid in the study of mongolism through more detailed and critical endocrine analyses and there has appeared a more favorable attitude toward intellectual and social development possibilities in the mongols and mongoloid types.

3) The neurosurgical approach to certain cases of mental defect due to developmental structural pathology in the central nervous system is giving rise to an improved point of view with regard to the successful relief of a limited number of cases of hydrocephaly, of porencephalic cyst, of certain types of craniostenosis, and of special types of epilepsy.

4) Among the endocrine disturbances associated with mental defect pubertus praecox (virilism) appears to be more frequently associated with adrenal cortical tumor or hyperplasia than with the pineal or other endocrine glands. Successful removal of adenomatous tumors of the adrenal has been known to reduce physical distortions and, by elimination of aggressive behavior affords some promise of educational accessibility. Cretinism, one of the severest *viable* forms of mental defect, shows appreciable response to correction by thyroid extract or thyroxine administration.

5) In mental defect due to infections of infancy and childhood improved techniques for the accurate diagnosis of congenital lues,

and advances in the methods of its treatment appear to indicate some improvement in its prognosis. The development and use of immune sera and of the sulfa drugs in the field of childhood infections may be expected to aid in the prevention of destructive organic consequences of severe infectious diseases.

6) Our better understanding of the importance of pre-natal trauma and maternal toxic states in the etiology of mental defect should lead to greater care in the management of pregnancy. It has been shown that fetal protection by placental resistance is not complete and that embryonic tissues are vulnerable to toxic agents which do not necessarily constitute any real threat to the maternal well-being. Toxic, anoxic, deficiency and infectious states which in the adult nervous system may be reversible or completely recoverable can produce permanent changes in the fetal neural structures leading to impairment of intellectual potentiality. Research in the subject of fetal and infantile anoxemia has been given great impetus in the past few years. The role of anoxic encephalopathy in the production of mental defect is a briskly debated subject. While a great deal has been learned regarding cellular physiology and chemistry under the influence of reduced tissue respiration its etiologic importance has not yet been fully evaluated.

7) Emotional factors, the effect of unfavorable environmental stresses and other indirect influences appear to play only a limited causative role in the production of mental defect.

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The History of Psychiatry

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Topeka, Kansas

Psychiatry, a specialized branch of medicine which has to do with the causes and treatment of abnormal individuals who are unable to fit into the routine of daily living, has taken many centuries to develop into the technical science it is today. Little by little, down through the ages, man has discovered more and more about himself and his behavior. In the beginning, when men noticed unusual actions of their fellow-men, they believed that they were possessed by demons. This central idea carried on, century after century, manifesting itself in the persecution of the insane until such people as William Tuke, Philippe Pinel, Jean Esquirol and Dorothea Dix began to throw off the chains of the mentally sick. It is primarily through the efforts of these and others that the care and treatment of the mentally ill has progressed to the point it is at the present time. In order to present a history of psychiatry in more simplified form, I have attempted to compile a brief history from numerous sources.

From the study of early man, it has been found that even he was not without mental disease of one kind or other. Primitive peoples came to believe, through their contact with nature, seeing the leaves of a tree move, hearing the water of a small brook rush over the rocks, that there were good spirits and bad spirits. When a river overflowed, killing many and destroying much, these people believed that the spirit of the river had been displeased, and that this was his way of retribution. Along the same line, the primitive believed that human behavior was motivated by good and bad spirits also. When abnormal actions were noted in an individual by his fellow-men, it was believed that because of his sins and wrong-doings this individual was thought to be "possessed" by one or more evil spirits. Early people and cultures interpreted evidences of mental disorder in different ways. The Bible speaks of King Saul being troubled by "an evil spirit of the Lord."[1] At one time, it was thought that a demon inhabited each of thirty-six parts of the body, each of whom must be kept constantly placated. When a person became "possessed," primitive logic immediately suggested that the evil-doing demon was showing his displeasure of the person's behavior. This demon, then, must be either placated or driven out of the individual by such extreme measures as flogging or starving. If, however, the actions and speech of the afflicted appeared to the populace to retain a religious or political bearing, the individual was held in high esteem.

Although this idea of demonology was general throughout the world of primitive thinking, as early as 860 B. C. there were beginnings of humane treatment in the Temples of Saturn and Aesculapius in Egypt and Greece, respectively, where those who were afflicted with a mental illness were treated by the priests, who made a study of such afflictions. The priests at these temples, which were usually situated in the mountains as near as possible to a medicinal spring, employed methods which at that time were revolutionary. They were kindness, suggestion, occupation, music and recreation. These features are today included in modern therapy.

Demonism persisted even after the birth of Christ, and all sorts of trickery were used to cast out these devils. Everyone, it was claimed, had 1,000 devils to the right of him, and 10,000 devils to the left. Gregory the Great, in 560 A. D., wrote of a nun who swallowed a bite of lettuce, without first making the sign of the cross, and consequently she swallowed a devil.

Hippocrates (460-370 B. C.), usually regarded as the father of medicine, was the first to discard the theory that diseases, both mental and physical, were of sacred origin, and believed that they were of natural causes, for instance, that melancholia was due to an excess of bile.

The school of physicians which followed Hippocrates carried on his teachings. They used hydro-therapy, massage, friction, gymnastics, and hypnotism. Rooms for excitable patients were supposed to be small, cool in summer, warm in winter, with walls that were smooth and mono-tone in color.

Plato (428-347 B. C.) and Aristotle (384-324 B. C.) were philosophers, and necessarily interested in the mental life of man. Both main-

tained but did not advance the understandings put forth by Hippocrates. Galen (131-201 A. D.) was very religious and more philosophical than Hippocrates, and certainly less factual. He did, however, describe much of the anatomy of the brain. Other physicians of the Roman era treated mental illness with bleeding, purging, and with sulphur baths.

As a whole, however, the treatment of mental diseases was limited primarily to incantations, sacrifice, prayers, and purification. The Greeks began bleeding and purging as therapy, and later supplemented this with dieting, exercise and education. They strongly advocated preventive measures for disease, and in general, reached the standard of development which was more progressive than anytime during the next fifteen hundred years.

During the centuries which followed, the attitude toward mental patients was that of fear and superstition, of ignorance and cruelty. This attitude was reflected in the barbaric treatment accorded them. This has been referred to as the "Period of Cruelty." Humane measures were almost unheard of, except in the monasteries, where patients with mild symptoms were received and given care. Those whose symptoms were more severe were thrown into dungeons, where they were flogged, fettered, starved and scourged, in the belief that the demons could be driven out by these means.

There were three methods used in casting out the devil. First, by a priest laying his hands upon the patient and repeating a prayer, for holy words were thought to be distasteful to the demons; second, by addressing the patient disrespectfully, attempting to disgust the demons or offend their pride; and third, as mentioned before, by punishing or torturing the patient. This latter method soon degenerated into the vile abuse of mental patients. Both pagan and Christian civilizations used herbs and drugs in exorcising the demons.

Wichcraft was much in evidence. Possession by a witch was diagnosed in much the same way we diagnose hysteria today—spots of anesthesia, seizures and trances. In Treves, a Prussian city of 49,000 population, within a space of a few years, 6,500 people were executed, found guilty of practicing witchcraft. Epilepsy was treated by burning a rooster alive, with a lock of the patient's hair and nail parings, where the patient fell in a seizure. Hallucinations were treated by feeding the patient raw wolf's flesh. Whipping, fetters, putting the patient in seclusion, herbs, emetics and purgatives were all supposed to rid him of excess liver bile, thought the cause of most mental diseases. Demons were also thought to lurk in the hair of the victim, so he was ducked under the water head first and held there, or ducked three times, bound, and left for the night. If, by morning, the patient had freed himself, he was "cured," but if he was still there, he was considered quite hopeless. Veins under the tongue were opened and blisters applied. "Backbones" were applied with a balm made of earthworms and bats. Mouth-gags and witch's bridles were used to hold in check noisy women.

Many mental cures were also used. Bloodroot, or a red juice, was used as a specific for blood diseases, yellow flowers or juices for a jaundiced condition. Walnuts were used for seizures, the meat of the nut representing the brain, inside of the shell, which represented the skull; the husk, the scalp, and each part of the nut was used for treatment of the part of the brain believed to be diseased.

Stones were considered beneficial. Topaz healed the person of his "Passion of Lunacy," the bezoar stone removed sadness and restored merriment to the afflicted. The onyx caused sleep.

In England in 1537, a house was granted to the corporation of London by Henry VIII for the confinement of fifty mental patients. This was called Bethlehem Asylum, later corrupted to Bedlam. Some of the patients who were not too violent were allowed to go out into the streets and beg for charity, and were popularly called "Bedlam beggars," and "Tom-o'-Bedlams."

> Bedlam beggars who with roaring voices . . .
> Sometimes with lunatic bans, sometimes with prayers,
> Enforce their charity.
>
> King Lear, II, 3.

The attendants were allowed to exhibit the patients for the fee of one penny. The annual income from this source reached four hundred pounds. The sanitary conditions in this and other homes established for the care of mental patients were deplorable. They were ill clad, naked or in rags, had no beds to sleep

in, only a bit of straw thrown on the floor. The most ingenious and incredible forms of torture were used, and their use was approved of by the physicians. Patients were whirled at a high rate of speed in a revolving machine. "Surprise" baths were employed; the patient was taken to his bath as usual and plunged into it all the way to the bottom of the tub in icy cold water and utter darkness. Other horrors which they had to endure were starvation, horrible noises and smells, and slimy dungeons with no light or ventilation, and as often as not infested with vermin. Many of these people were forced to wear heavy iron collars, belts and anklets, by which they were chained to the wall.

There were, however, bright spots in this tragic situation. At the beginning of the fifteenth century, a colony of mentally sick people was formed at Gheel, Belgium, where St. Dymphna was buried. This was the only colony of its type. The patients were free to live with inhabitants of the village, and to carry on a normal existence. In France, in 1792, Philippe Pinel was appointed physician to Bicetre, the great hospital for male patients in Paris. As conditions in France were as bad as in England, he set about to change and improve them with a policy known as the open door method.

"At the risk of his own life and liberty, he initiated the reform of striking off their chains, placing them in hospitals under lenient physicians and doing away with the abuses of drugging and bloodletting to which they were subjected."[2] The removal of the chains of the insane by Pinel was made the subject of a painting by Tony Robert Fleury. It is one of the celebrated pictures of the world, and now hangs in the lecture room of the Salpetriere Hospital in Paris.

Simultaneously with Pinel, a non-medical man, William Tuke, in 1794, raised funds for what was called a "retreat" for the Friends' Society in England, in which all restraint was abandoned, and sympathetic and understanding care with occupational facilities were provided. This was much the same idea as that of Pinel's open door method.

Jean Etienne Dominique Esquirel, a pupil of Pinel, in 1810, successfully instituted changes in the housing for the insane in France. He established ten new asylums patterned on Pinel's open door method, and he was the first to lecture publicly, in 1817, on psychiatry.

In 1784, the Lunatick Tower in Vienna began a discreditable "career" which lasted until 1853. It was an ornately decorated round tower, within which were square rooms. The doctors and "keepers" lived in the square rooms, while the patients were confined in the spaces between the walls of the square and round parts.

During the eighteenth century, many mental hospitals were initiated. The first epileptic hospital was founded in Alsace, France, in 1486, but at that time the disease was thought to be contagious. Mental asylums, as they were called, began with Bethlehem in 1537; St. Luke's in England in 1751; the Pennsylvania Hospital in Philadelphia in 1752, at which Benjamin Rush, the "father of American psychiatry," entered upon his duties in 1783; the famous psychiatric hospital in Moscow in 1764, and the still standing Narrenurm in Vienna in 1784.

Williamsburg, Virginia, was the site of the first hospital in the United States built exclusively for mental patients. It was constructed in 1773, and is now known as the Eastern State Hospital. The Friends Hospital at Frankford, Pennsylvania; McLean Hospital at Sommerville, Massachusetts, and Bloomingdale Hospital in New York were some of the first mental hospitals to be established in the United States, but a Senate report of 1829 showed them to be little more than repositories.

One name, however, will forever be associated with the movement for the proper housing, care and treatment of the mentally ill in the United States. In 1841, Dorothea Lynde Dix made an investigation of an asylum in Massachusetts, and she became so aroused that she presented to the Legislature of the State a long report or "Memorial," demanding that proper housing and care be given these poor unfortunates. Nor did she stop here. She expended tremendous efforts, traveling about this country and Europe, lecturing, organizing and pleading for appropriations for the construction of mental hospitals. Her efforts alone were responsible for the establishment of thirty-two institutions.

Formal research into the causes and nature of nervous and mental diseases gained headway under the inspiration of Jean Charcot a great French neurologist, who contributed to

psychiatry by his description of hysteria as a physical representation of an idea.

Probably the most outstanding contribution to psychiatry has been that of Sigmund Freud (1865-1939) through what is known as psychoanalysis. Offshoots from Freud's original school of psychoanalysis have been numerous. The most important of these are Carl Jung's "Analytical Psychology" and Alfred Adler's "Individual Psychology."

In addition to psychoanalysis, another point of view began in this era. The Mental Hygiene Movement developed through the efforts of Clifford Beers, a layman who, in 1908, published an autobiography, "A Mind That Found Itself." This book tells of his own experience as a psychiatric patient in several mental hospitals. By enlisting the aid of psychiatrists, lawyers, clergymen and other outstanding citizens, he began first a local Mental Hygiene Movement, and then a National Movement, which has since become international. The Mental Hygiene Movement has been followed by the Child Guidance Movement, by the spreading of psychiatry into industry, criminal and juvenile courts, and into penal institutions.

Looking back over the ages, we can see four major trends develop in medicine, particularly in relation to psychiatry. First, the period in which illnesses of any kind were not clearly understood, and the physician-priest-magicians were so concerned with driving away bad demons that their treatment degenerated into *persecution* of the sick.

Persecution reached its climax with the burning of witches, and was stopped later by the intervention of the church which became a sanctuary for the sick and helpless. This began the second trend of medicine, resulting in little more than *segregation* of the sick, and marking the beginning of hospitals and asylums.

As the practice of medicine became defined, it became more a true science, educating society to pity rather than to condemn the sick, and at the end of the eighteen century, the *humanitarian* attitude toward the sick indicated the third trend in medicine.

By the beginning of the twentieth century, medical science had advanced far enough to bring about a fourth trend—*prevention* of illness. During this period, Mental Hygiene Movements started, nationally and internationally.

In the last two decades, successful attacks upon mental disorders were made by special physical remedies, even though psychoanalytic medicine still enjoys a huge following. Outstanding is the introduction of malaria and other types of fever therapy in 1917 by Wagner-Jauregg, which is now indispensable in treating neurosyphilis and chorea; the use of sodium amytal to produce prolonged sleep; and the more recent and amazing shock methods of treating schizophrenia with insulin as introduced by Sakel, and the gratifying results in the treatment of mental depressives by the convulsion-producing drug known as metrazol.

Down through the ages, the science of psychiatry has been progressing steadily until today mental disease has been put on an equal footing with other forms of illness, and now receives the same intensive study and skillful nursing as does any other problem in general medicine.

In 1928, the number of insane in the United States had been reduced by 3,391, compared with 1923, and what is more important, a reduction in the rate per 100,000, over that of 1923, from 245.0 of 1923 to 221.4 in 1928.

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A Further Report on the Use of Shock Therapy

Results in 1302 Cases

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In September, 1940, we reported[1] on the first 500 cases treated with shock therapy at the New Jersey State Hospital at Trenton. Since that time the number has increased to 1,302. Our method and technique of treatment is still the same as noted in the previous paper, except that electric shock has been substituted for metrazol. Briefly, our principal form of shock treatment is insulin. This is given to all types of functional psychoses and neuroses. If after 20 insulin treatments the patient has not shown decided improvement, electric shock is added to the treatment in the third hour of the insulin reaction. The exception to this rule is manics, markedly agitated depressions and catatonics. With these patients, we start the electric and insulin concurrently. We would like to emphasize the importance of producing a real shock and we endeavor to give insulin in sufficient doses to produce a defenseless coma and electric shock is used to produce a definite epileptiform convulsion. We believe that many of the unfavorable end results from the use of shock therapy are due to the failure to carry the treatments to the extent that we believe necessary and to which we subject our patients.

We have always believed that the only criterion worthy of consideration was whether the patient was in condition to be returned to the community and have always evaluated our results on this ability. Of our 1,302 patients, 25 died, 43 were terminated because of physical conditions, leaving a total of 1,234 who completed a course of treatment. Of this 1,234, 919 were released from the hospital or 70.6 per cent. Of these, 134 returned. These were subject to further treatment and 51 of this number were again released.

On July 1, 1943, we had 836 patients, or 64.2 per cent, out of the hospital. The following table shows the diagnostic distribution of these patients and their status as of July 1, 1943:

After six years of using shock therapy, we still are of the opinion that at this time shock therapy offers the best method for a rapid improvement with a higher percentage of release for the functional psychoses than any other method yet devised.

Reference

1 Taylor, John H.: "Results in the First 500 Functional Cases Treated with Insulin Therapy and Its Modification, at the New Jersey State Hospital, Trenton," *Dis. Nerv. Sys.*, Vol. 1, No. 9, (Sept.) 1940.

	Total	*Released*	*Ret. & Released*	*Returned*	*Died*	*Stopped*	*Never Released*
Manic Depressive	274	232	30	52	14	18	212
Schizophrenia	682	438	15	14	5	7	30
Involutional Psychosis	124	91	3	8	4	11	18
Psychoneurosis	65	57	2	2	0	1	7
Other Types	157	101	1	7	2	6	48
Total	1302	919	51	83	25	43	315

Neurological Clinical Pathological Conference

of the

Cincinnati General Hospital

CHARLES D. ARING, M.D., *Editor*

CASE 152121

Presentation of Case

A 59-year-old white man was admitted to the Neurological Service on November 28. On November 21 he suffered the onset of what his physician termed "flu," characterized by generalized aches and pains, particularly in the back. He was put to bed and his temperature was found to be 104 degrees F. The practitioner administered sulfapyridine on November 23, and after the patient had taken about six grams, his medication was changed to sulfamethylthiazol because of nausea. He tolerated the latter drug well, improved; and was able to be up for meals with his family.

The back pain had persisted, though it was not incapacitating, and on noon of November 27 he had more pain between the scapulae than anywhere else. On that day after the evening meal he found that he could not arise from the table; he had to be helped to bed. He noted numbness of the lower extremities and inability to use them. On November 28 he was admitted to the hospital because of progression of all symptoms.

The past history contained several points of possible significance. Fifteen years previously he had had "double pneumonia" and had retained a cough up to the time of his death. Six months before he had had the right eye enucleated for chronic glaucoma. Two months prior to death his physician told him that he had Bright's disease; he had had all the teeth removed one month later on this physician's advice.

Examination and Course—On admission the temperature was 100.4° F. rectally, the pulse rate 124, and the respirations 24. The patient was alert and cooperative. At 2:30 P. M., he was unable to perceive touch, pain, or temperature below D-12; deep pressure and passive changes in position of the toes were appreciated. The plantar responses were weak but definitely flexor.

By 5 P. M. the level had ascended to D-6, below which nothing was sensed. He was unable to sit up unassisted, and no movement was observed in the lower extremities which were flaccid. Tendon reflexes were absent. There was no response to plantar stimulation. Tendon reflexes in the upper extremities were normal. The left corneal reflex was normal, the right eye had been removed. The bladder was distended; catheterization was performed and tidal drainage of the bladder instituted. Seven hundred cc. of urine were obtained at the initial catheterization. The urine had an offensive odor. The abdomen was distended with gas.

At 6 P. M. he became dyspneic. His pulse was weak and he perspired freely. He took fluids well and sulfapyridine was begun by mouth. He expectorated thick purulent sputum. The respirations became labored and involved the use of accessory muscles. At 10 P. M. an observer noted that he was unable to move the right upper extremity. He appeared to rest quietly through the night, though he was weak and listless. On the morning of November 29 he was pale and gasped for breath. He died at 10:45 A. M., after a hospital course of 20 hours.

Laboratory Data—The temperature remained around 100 degrees F. (rectally) and the pulse rate about 120; the respirations rose gradually to 35 per minute. The blood pressure was 85/50 on admission and 50/40 five hours later. The red blood count was 5.1 million, the hemoglobin 15 grams, the white blood count 29,300. In the differential count, there were 91 per cent neutrophils and 9 per cent lymphocytes. The specific gravity of the urine was 1.015 and the only abnormality disclosed in the urinalysis was a heavy cloud of albumin. The blood urea nitrogen was 75 mg. per cent. The blood Kahn was negative. Roentgen plates of the chest showed active and chronic bronchitis.

An electrocardiogram revealed severe myocardial damage. The QRS interval was prolonged. T-1 and 2 and 4 were upright, T-3 inverted; ST segments 1 and 3 were elevated,

2 depressed. Intraventricular block was interpreted.

Lumbar and cistern punctures were done immediately after death (as the patient ceased to breathe). The cerebrospinal fluid from the lumbar sac was slightly yellow and contained 5 white blood cells per cmm. The protein content was 330 mg. per cent, the colloidal gold curve and Wassermann were negative. The cistern fluid resembled water, contained 6 white cells per cmm., and the protein content was 14 mg. per cent.

Differential Diagnosis

Dr. Milton Rosenbaum—I think the differential diagnosis is between two diseases, epidural abscess and some type of acute myelitis of the ascending type. For the sake of completeness, one would think of neoplasm at the age of 59; a patient who has a malignant tumor with metastases to the cord may suffer a rapid onset. If the vertebrae are involved and the metastases are around the cord, a vessel may be occluded and there results an acute myelitis on a vascular basis. The total picture in this case is against that; the process obviously is an infectious one.

The process was an acute one, and the lack of certain expected neurological signs may be explained on the basis of spinal shock—the failure to obtain an extensor plantar response, for example. The urinary retention and the low and falling blood pressure favor this idea.

An important point was pain in the back which was severe, a symptom classically seen in acute epidural abscess. It may be seen in myelitis but it is not a presenting symptom. There is every evidence that this man was suffering from root symptoms.

In acute myelitis or epidural abscess, an acute onset may occur. There was certain evidence that the process ascended. Does this mean that something started lower down in the cord and gradually worked up? Or does it mean that something started higher and gradually compressed the spinal cord? As far as epidural abscess is concerned, I think one may see either type of involvement. The abscess may spread through the epidural space; if the abscess spreads upward, the sensory level extends upward. I would think in most cases it is more common to have epidural abscess spread rather than remain localized.

No mention is made of point tenderness, that is, local pain over a single spinous process or two, a finding that would be practically pathognomonic of epidural abscess, if it was associated with the other symptoms from which this man suffered. If this maneuver had been done in this case, and tenderness found over a vertebra, that would have practically proved the diagnosis of epidural abscess. Then the next thing to have done would have been to insert a spinal needle gently, aspirating occasionally in the attempt to tap pus. This must be done carefully, for if pus is transferred into the subarachnoid space, meningitis will result.

The usual epidural abscess begins with pain in the back followed by weakness, and by evidence of staphylococcus infection. The infection usually is blood borne, and it reaches the epidural space via the blood stream or through the intervertebral foramina as the result of direct extension from osteomyelitis. In this case we have a history of chronic infection of the lungs; possibly extension occurred from the lungs. The extraction of teeth may have stirred up latent infection and transmitted it via the blood stream. The fact that this patient's disease was not cured with sulfonamides may indicate that the staphylococcus was the offender.

An important point is the lumbar and cistern fluids which were obtained at the time of death. There was 330 mg. per cent protein in the CSF from the lumbar sac and 14 mg. per cent protein in the cistern. No dynamics were obtainable. It is assumed that this man had a subarachnoid block. I think the fact that the cistern fluid was so normal indicates that the block was low down. If this patient had a block of the spinal canal, myelitis is practically ruled out by the rarity of subarachnoid block in this condition, and the diagnosis of epidural abscess remains.

Why did death occur so quickly if the patient had an epidural abscess? One is apt to forget that the disease was fatal within a week, and possibly within two days. It may be that he died quickly because of ascending infection. The process may have been localized in the epidural space about the cervical cord, with resultant edema of the cord and involvement of the nerve supply to the respiratory muscles. The general physical condition was not too good, he had bronchitis, an ailing heart, and Bright's disease.

Student—How long should it take to develop a protein content of this magnitude in the lumbar sac?

Dr. Rosenbaum—In cases of epidural abscess in which the onset is rapid, the protein may be as high as 200 mg. per cent within a week of the onset. It is not only the block that is functioning in this respect, the infection in the epidural space may have something to do with stasis, as far as absorption of CSF from the lower subarachnoid space is concerned.

Dr. Joseph P. Evans—At operation occasionally there is, in processes of such clinical acuteness, obvious evidence that the infection has been of long standing and has been lying latent. The first epidural abscess described showed chronic granulation tissue in the epidural space. In my own experience in cases of acute onset, at operation there is chronic granulation tissue. I think the process occasionally may have started considerably earlier than the overt symptoms indicate and partial block may have resulted, and then, for some reason, acute flare-up occurs and causes transection.

Other points are that one mustn't expect roentgen changes in these cases since it takes weeks for them to appear; also that there may be epidural infection associated with thrombosis of veins, but then there are others in which there is a primary osteomyelitis.

The rate of recovery from epidural abscess is in direct proportion to the promptness of surgical drainage. It should be possible to make the diagnosis before paralysis develops. If paralysis has supervened, the rate of recovery is slow.

Clinical Diagnosis

Ascending thrombosis of anterior spinal artery.

Dr. Rosenbaum's Diagnosis

Epidural abscess.

Anatomical Diagnosis

Visceral and nervous lesions characteristic of infectious polyneuritis (infective neuronitis, polyneuritis with facial diplegia, Guillain-Barré syndrome).

Acute and chronic bronchitis.

Acute and chronic pyelonephritis with abscess formation.

Arteriolonephrosclerosis.

Generalized arteriosclerosis.

Pathological Discussion

Dr. I. Mark Scheinker—The gross examination of the brain and spinal cord revealed no changes. There was no block within the spinal column; the subarachnoid spaces were free, the leptomeninges clear and uninflamed.

The routine stains of the spinal cord did not reveal much. The leptomeninges were normal and the same appeared to be true of the parenchyma of the spinal cord. In myelin sheath stains of the spinal cord there was some rarefaction with vacuole formation in the dorsal column. The main tracts were well preserved and the subarachnoid space and leptomeninges were normal. In gold and silver impregnations the axis cylinders in the dorsal columns were considerably changed.

A superficial examination of this spinal cord would have revealed nothing particularly abnormal. Undoubtedly just such superficial examinations have resulted in the confusion which has surrounded similar cases.

In the spinal roots the axis cylinders were enormously swollen and partially degenerated. Also in the posterior columns tremendous swelling of the axis cylinders could be demonstrated with the proper stains. There was no inflammatory change and no vascular change. It is interesting to note that the myelin sheaths of the posterior columns were well preserved, though they suffered secondary compression apparently due to the tremendous swelling of the axis cylinders. The rings of myelin were dilated and irregular in outline.

In the kidneys there were diffusely scattered, miliary abscesses and an obvious interstitial nephritis. There had occurred an infiltration of cells, particularly lymphocytes in the connective tissue capsules and spaces of the liver. There were focal accumulations of round cells in the adrenal glands. The liver parenchyma showed some slight focal vacuolation. The heart contained areas in which the muscle fibers appeared frayed and in some instances vacuolated. There were occasional, small collections of round cells between the heart muscle fibers.

In this case the main alterations were found in the spinal cord roots, and less in the axis cylinders of some of the white matter of the

cord. The change consisted of a primary swelling of nerve fibers unassociated with inflammatory changes or vascular disturbance. The final diagnosis was infectious polyneuritis.

Does this pathology help to elucidate some of the clinical findings of infectious polyneuritis? A feature of infectious polyneuritis is that if improvement begins, progress thereafter is considerably more rapid than in other forms of polyneuritis. If the change in the nervous system consists primarily in swelling of the axis cylinders, particularly in the roots and peripheral nerves, there is always the possibility of recovery.

Another characteristic feature of infectious polyneuritis is the increased protein content of the CSF. The pathology of infectious polyneuritis may help to explain the characteristic CSF. There is clinical and experimental evidence that some absorption of CSF occurs via the perineural spaces; some support is lent this idea by the relatively large accumulation of tumor cells that may be found around nerve roots as they pierce the dura mater in those persons who have leptomeningitic carcinomatosis or sarcomatosis. Also, the absorption of dye (methylene blue) from the subarachnoid space surrounding the cord takes place chiefly along the perineural spaces as has been demonstrated by Spatz.

If it is true that the main pathology of infectious polyneuritis is swelling of axis cylinders, then there is an increase in volume of spinal roots. A block to absorption may occur in this manner, but the block is between the subarachnoid space and the perineural spaces due to the occlusion of these openings. It is possible that this mechanical occlusion resulting in stagnation is responsible for some of the protein increase in infectious polyneuritis. This thesis comes a cropper when we think of the tremendous protein increase that may occur in the CSF in the spinal block of cord tumor. Possibly the absorptive power in this area is related somehow to the protein content of the fluid.

The facial nerve is the cranial nerve most frequently involved in infectious polyneuritis. The only cranial nerve that passes through a long, narrow canal is the facial nerve and the same type of mechanical interference may be responsible for its rather frequent involvement in this disease.

The interesting difference in the protein content of the cistern and lumbar fluids in infectious polyneuritis might be due to the fact that the disease is so often much more severe in the spinal cord than in the brain stem, and the stagnation of CSF due to lack of absorption is so much greater in the spinal canal.

Dr. Charles D. Aring—It is fair to say that had the dynamics of the CSF been known, and they should have been, the correct diagnosis would have been suspected by Doctor Rosenbaum. A most significant point was the rapid ascent of nervous symptoms and the final involvement of the high cervical cord (weak arm, respiratory distress). Epidural abscesses usually occur in the dorsal region where the epidural space is relatively large, though they are found occasionally in the cervical region. Death supervened much too rapidly here to be accounted for by an epidural abscess. If one had to explain the paraplegia, incontinence, myocardial damage, falling blood pressure, and excess protein in the lumbar CSF all on one basis, it most likely would have to be a condition like infectious polyneuritis or myelitis.

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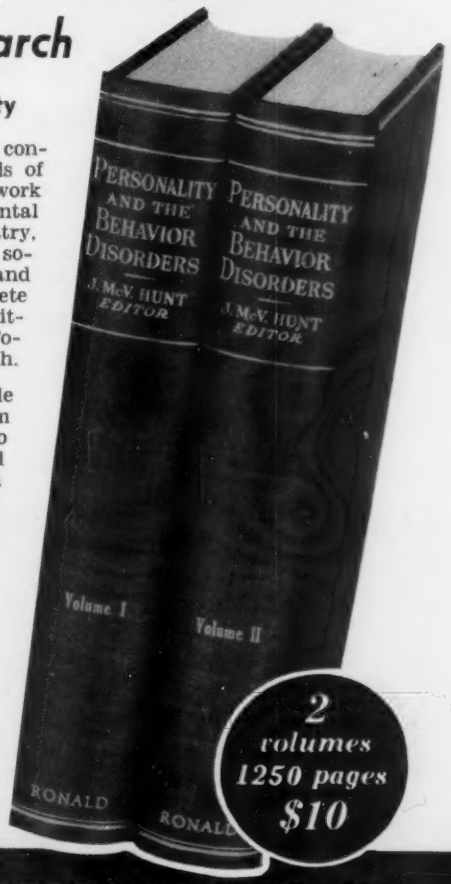

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BOOK REVIEW

THE TECHNIQUE OF SELF-HELP IN PSYCHIATRIC AFTER-CARE: Vol. 1, The Historical Development of Recovery's Self-Help Project by A. A. Low, M.D.; Vol. 2, Group Psychotherapy by A. A. Low, M.D.; Vol. 3, Lectures to Relatives of Former Patients by A. A. Low, M.D. Publications of Recovery, Inc., Chicago, Illinois.

The above three booklets were written by the founder and president of the now liquidated Recovery, Association of Former Patients. "The Historical Development of Recovery's Self-Help" is a vivid account of the life of the project. Founded at the time of highest expectations for the various shock treatments, it parallels in its curriculum the waxing and waning enthusiasm toward the treatments which gave birth to it. It depicts the fight against the "stigma" of mental illness, the attempts of improving commitment procedures and the mutual help, often coupled with business acumen, of former patients. "Group Psychotherapy" illustrates the author's approach by giving more or less complete shorthand transcripts of actual sessions. Both the single interview and the multiple interview methods are presented. The latter is now the preferred method of the author. The so-called contrast formulation technique in which "local sensations" are "contrasted with generalized sensations, normal interpretations with abnormal delusions, false beliefs with acceptable ideas, danger with distress . . ." and short theoretical explanations are skillfully blended in dealing with the problems presented by the patients. "Lectures to Relatives of Former Patients" probably is the most ambitious attempt to assist in bringing about those environmental conditions most conducive in preventing relapse. The author's ingenuity in discussing the various topics is beyond question, although one may not always agree with some rather theoretical presentations and formulations.

After reading the booklets one cannot feel anything but regret for the discontinuance of the project. It seemed to be a positive step for following up the immediate results of the shock treatments by psychological means, and one's interest is aroused in what the ultimate result of such an approach might have been in preventing relapses. After all, the success or failure of such an undertaking is measured by its actual accomplishment rather than by the strictly academic contents of its teachings. It would be, therefore, of interest to have comparable statistics on that score.

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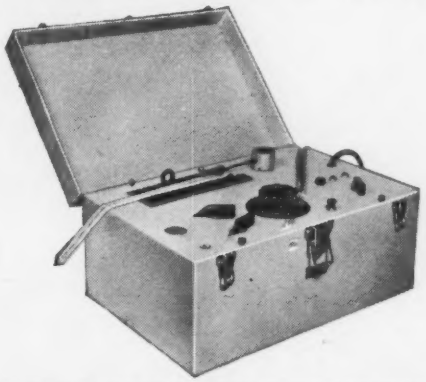

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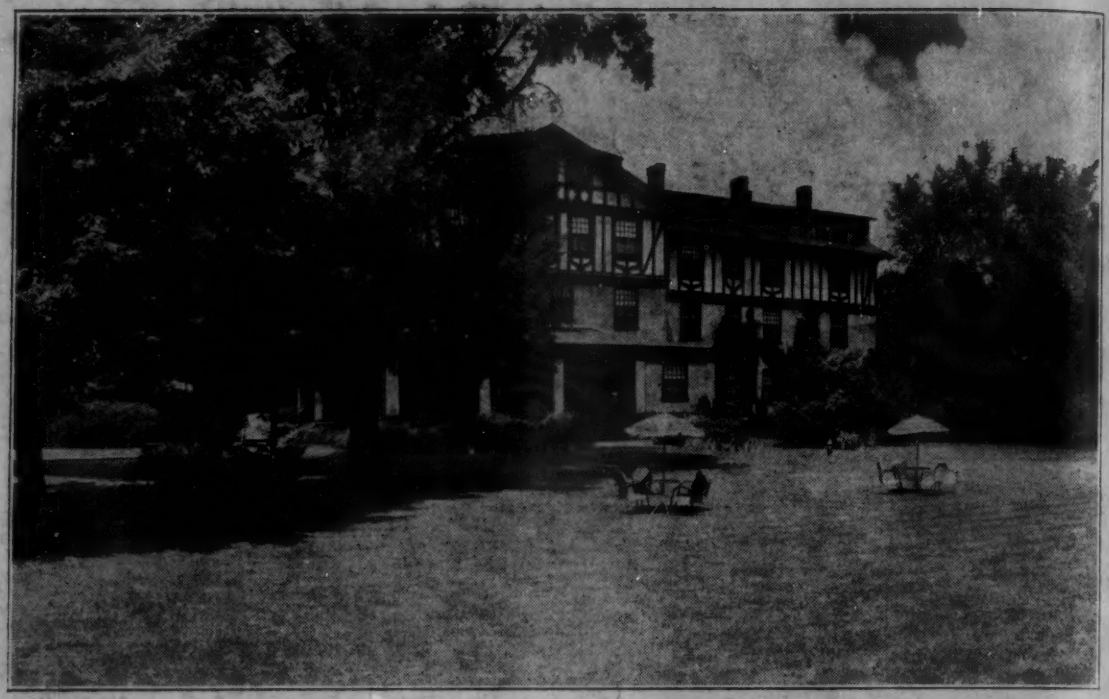

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